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Issue Date: 22 June 2007

In the Matter of

Mr. C.K.,[1]
Claimant

Case No.: 2004 BLA 5465

v.

BEATRICE POCAHONTAS COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

Appearances: Mr. Ron Carson, Personal Representative
For the Claimant

Mr. Douglas A. Smoot, Attorney
Ms. Ashley M. Harman, Attorney (on brief)
For the Employer

Before: Richard T. Stansell-Gamm
Administrative Law Judge

**DECISION AND ORDER ON REMAND –
DENIAL OF BENEFITS**

This matter involves a claim filed by Mr. C.K. for disability benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"), as implemented by 20 C.F.R. Parts 718 and 725. Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

[1]Chief Administrative Law Judge John Vittone has directed that I substitute initials for the names of the Claimant and all family members. Any comments or concerns regarding this mandated practice should be directed to Chief Administrative Law Judge John Vittone, 800 K Street, Suite 400N, Washington, D.C. 20001.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Remand Determination[2]

On June 29, 2005, I issued a Decision and Order denying Mr. K.'s claim for black lung disability benefits. The essential inquiry before me was whether Mr. K. had established, under 20 C.F.R. § 725.309(d), a change in a condition of entitlement since the denial of his most recent prior claim by establishing his total disability was due to coal workers' pneumoconiosis. Based on the more probative opinions of Dr. Castle and Dr. Rosenberg, I concluded that Mr. K. failed to establish that his total respiratory impairment was due to coal workers' pneumoconiosis. Mr. K. appealed and on April 19, 2006, the Benefits Review Board ("BRB" and "Board") remanded his case with directions that the medical opinions of Dr. Rosenberg and Dr. Castle be re-evaluated in light of the decisions in *Scott v. Mason Coal Co.,* 289 F.3d 263 (4th Cir. 2002) and *Toler v. Eastern Associated Coal Corp.,* 43 F.3d 109 (4th Cir. 1995).

Background

In Mr. K.'s most recent prior claim,[3] Administrate Law Judge Nicholas Laezza in 1989 determined the preponderance of the chest x-rays in the record at that time established the presence of pneumoconiosis. Additionally, the preponderance of the medical opinion supported a finding of clinical pneumoconiosis. Subsequently, the BRB affirmed the Judge Laezza's finding of coal workers' pneumoconiosis. Additionally, during the 1996 adjudication of Mr. K.'s modification request, Administrative Law Judge Robert Kaplan concluded the preponderance of the more recent arterial blood gas studies demonstrated that Mr. K. had a totally disabling respiratory impairment. However, Judge Kaplan denied the modification request and claim because Mr. K. failed to establish that his total pulmonary disability was due to coal workers' pneumoconiosis.

As a result, in the record before me as part of Mr. K.'s third, and subsequent, claim, five doctors considered whether coal workers' pneumoconiosis caused Mr. K.'s total disability. Due to insufficient documentation and explanation, I concluded the assessments of Dr. Forehand and Dr. Thakkar suffered a loss of probative value. The remaining three physicians rendered documented and reasoned, though conflicting, causation opinions. A treating physician, Dr. Smiddy, concluded both cigarette smoking and exposure to coal mine dust contributed to Mr. K.'s totally disabling pulmonary impairment. In contrast, Dr. Castle and Dr. Rosenberg concluded that Mr. K.'s breathing difficulties were attributable to idiopathic pulmonary fibrosis. Both Dr. Rosenberg and Dr. Castle noted insufficient radiographic and clinical evidence of coal workers' pneumoconiosis. In evaluating these three opinions, I gave greater probative weight to the assessments of Dr. Castle and Dr. Rosenberg, based on their status as board certified pulmonologists and well integrated analysis. Accordingly, I concluded the preponderance of the more probative medical opinion established that Mr. K. was not totally disabled due to coal workers' pneumoconiosis.

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[2]As part of my assessment, I have considered the parties' briefs on remand.

[3]The extensive procedural history is set out in my June 29, 2005 decision.

Discussion

As noted by the BRB, *Scott* has a significant effect on my probative value assessment. The court in *Scott* set out two considerations for an administrative law judge presented with a medical opinion on the causation of total disability containing a conclusion that the miner does not have coal workers' pneumoconiosis which is contrary to the administrative law judge's determination that a miner has clinical pneumoconiosis.[4] First, citing *Toler,* 43 F.3d at 115, the *Scott* court reiterated that in a case where both coal workers' pneumoconiosis and total disability have been established, an administrative law judge may not credit a medical opinion holding the total disability is not due to coal workers' pneumoconiosis, unless he identifies "specific and persuasive" reasons for finding the doctor's determination does not rest on his disagreement that the miner does not have coal workers' pneumoconiosis. *Scott,* 289 F3d at 269. Second, in the event an administrative law judge provides the requisite reasons, the medical opinion would nevertheless "carry little weight, at the most." *Id.*

In light of these two *Scott* considerations, I conclude that at the opinions of Dr. Castle and Dr. Rosenberg on the causation of Mr. K.'s total disability have at best "little" probative weight. Although the extensive review of the record and well integrated diagnosis of idiopathic pulmonary fibrosis by both physicians precludes the automatic exclusion of their opinions on the causation issue, *Scott* emphasizes that even if such opinions are considered, they have little probative weight. Consequently, on remand, the probative weight balance has shifted significantly in Mr. K.'s favor. Due to the "little" probative value that may be give to the opinions of Dr. Castle and Dr. Rosenberg on the total disability causation issue, the remaining viable opinion of Dr. Smiddy that Mr. K.'s total disability is due to coal workers' pneumoconiosis has become the more probative assessment.

Based on Dr. Smiddy's more probative medical opinion, I conclude that Mr. K. has established total disability due to coal workers' pneumoconiosis, which in turn means Mr. K. has now proven an applicable condition of entitlement previously adjudicated against him as required by 20 C.F.R. § 725.309(d). As a result, I will review the entire record to determine whether he is entitled to black lung disability benefits. At this point, according to 20 C.F.R. § 725.309(d)(4), "no findings made in connection with the prior claim . . . shall be binding on any party in the adjudication of the subsequent claim" (emphasis added).[5] In other words, Mr. K. must once again prove all four elements of entitlement.

[4]Contrary to the assertion by Employer's counsel in the remand brief, the distinction based on an administrative law judge's finding of legal pneumoconiosis and a physician's conclusion that only clinical pneumoconiosis is not present, as noted by the *Scott* court in *Hobbs v. Clinchfield Coal Co.,* 45 F.3d 819 (4th Cir. 1995) (*Hobbs II*) and *Dehue Coal Co. v. Ballard,* 65 F.3d 1189 (4th Cir. 1995), is not applicable in Mr. K.'s claim. Unlike the physicians in *Hobbs* and *Dehue,* Dr. Castle and Dr. Rosenberg concluded Mr. K. had neither clinical nor legal pneumoconiosis. In that situation, the court indicated *Hobbs* and *Dehue* do not apply. *Scott*, 289 F.3d at 269.

[5]The two exceptions to this rule are not applicable in this case. The parties have never stipulated to the presence of pneumoconiosis and, on June 21, 1984, upon first notification of an initial determination of benefits for Mr. K., the Employer contested all elements of entitlement including the presence of pneumoconiosis (DX 2).

Stipulations of Fact

At the hearing, the parties stipulated to the following facts: a) Mr. K.'s length of coal mine employment was at least 30 years; b) Beatrice Pocahontas Company is the responsible operator in this case; and, c) Mrs. B.L.K. is an eligible spouse for the purposes of augmenting any benefits that may be payable under the Act (TR, pages 8 to 12).

Preliminary Findings

Born on April 27, 1929, Mr. K. married Mrs. B.L.K. on July 1, 1959. Mr. K. started mining coal in 1947. From 1950 until he stopped working in the coal mines in 1983, Mr. K. worked for the Employer. At the end of his coal mine employment, Mr. K. was a section foreman. In this position, Mr. K. helped the men he supervised timber the roof and complete other tasks they were assigned in the coal mine. The coal seams were 5 ½ feet high and the timbers weighed between 60 and 80 pounds. Mr. K. also took gas readings every two hours and walked with a slight bend through most of the day because of the low ceilings. The job required physical exertion and heavy lifting. All of his coal mine employment was underground and most if it was at the face of the mine. After a serious mining accident in 1980, which broke Mr. K.'s nose, damaged his teeth and injured his back, Mr. K. eventually stopped mining coal in 1983 due to breathing problems and with the advice of his physician. (DX 3, TR, pages 26 to 31)

Mr. K. is treated for his breathing and heart problems by Dr. Thakkar with medication. He is on continuous oxygen therapy. Mr. K. smoked cigarettes for 20 to 25 years, starting in his 20's and smoking a ½ pack to a pack of cigarettes per day until 1989 (TR, pages 31 to 33).

Entitlement to Benefits

To establish entitlement to disability benefits under the Act, a claimant must prove four basic conditions by a preponderance of the evidence. First, the miner must establish the presence of pneumoconiosis.[6] Second, if a determination has been made that a miner has pneumoconiosis, it must be determined whether the miner's pneumoconiosis arose, at least in part, out of coal mine employment.[7] Third, the miner has to demonstrate he is totally disabled.[8] And fourth, the miner must prove the total disability is due to pneumoconiosis.[9]

Pneumoconiosis

"Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment.[10] The regulatory definitions include both clinical, or medical pneumoconiosis,

[6] 20 C.F.R. § 718.202.

[7] 20 C.F.R. § 718.203(a).

[8] 20 C.F.R. § 718.204(b).

[9] 20 C.F.R. § 718.204(a).

[10] 20 C.F.R. § 718.201(a).

defined as diseases recognized by the medical community as pneumoconiosis, and legal pneumoconiosis, defined as "any chronic lung disease arising out of coal mine employment."[11] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment." 20 C.F.R. § 718.201(b). As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

According to 20 C.F.R. § 718.202, the existence of pneumoconiosis may be established by four methods: chest x-rays (§ 718.202(a)(1)), autopsy or biopsy report (§ 718.202(a)(2)), regulatory presumption (§ 718.202(a)(3)),[12] and medical opinion (§ 718.202(a)(4)). Since the record does not contain evidence that Mr. K. has complicated pneumoconiosis, and he filed his most recent claim after January 1, 1982, a regulatory presumption of pneumoconiosis is not applicable. In addition, he has not submitted a biopsy report and the record obviously does not contain an autopsy report. As a result, Mr. K. will have to rely on chest x-rays or medical opinion to establish the presence of pneumoconiosis. Additionally, under the guidance of *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), since Mr. K. last mined coal in Virginia, I must consider the chest x-ray evidence and medical opinion together to determine whether Mr. K. can establish the presence of pneumoconiosis in his lungs.

Chest X-Rays

Before I consider the various interpretations of the chest x-rays in this claim, I must address the manner in which I will treat the comments on the x-ray forms and treatment record radiographic studies.

While indicating that an administrative law judge may consider an x-ray reading with a profusion level of 1/0 or greater as positive for pneumoconiosis, in *Cranor v. Peabody Coal Co.*, 22 B.L.R. 1-1, 1-4 (1999) (en banc on recon.), the BRB discussed at least two types of specific comments that an interpreting physician might make along with a profusion of 1/0 or greater.

First, after finding a profusion of 1 or greater, a physician might also comment that another disease cannot be ruled out, as in *Melnick v. Consolidation Coal Co.*, 16 B.L.R. 1-31 (1991) (en banc). In this situation, the BRB concluded the physician's comment calls his diagnosis of pneumoconiosis based on profusion into question. *Id*. at 1-37. Consequently, that type of comment should be evaluated within the 20 C.F.R. § 718.202(a)(1) analysis about the presence of pneumoconiosis. If the comment suggests an alternative diagnosis, the "internal inconsistencies" between a diagnosis of pneumoconiosis due to sufficient profusion and an

[11]20 C.F.R. § 718.201(a)(1) and (2).

[12]If any of the following presumptions are applicable, then under 20 C.F.R. § 718.202(a)(3), a miner is presumed to have suffered from pneumoconiosis: 20 C.F.R. § 718.304 (if complicated pneumoconiosis is present, then there is an irrebuttable presumption that the miner is totally disabled due to pneumoconiosis); 20 C.F.R. § 718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more coal mine employment, there is a rebuttable presumption that total disability is due to pneumoconiosis); and 20 C.F.R. § 718.306 (a presumption when a survivor files a claim prior to June 30, 1982).

alternative diagnosis may "detract from the credibility of the x-ray interpretation under 20 C.F.R. § 718.202(a)(1)." *Cranor*, 22 B.L.R. at 1-5 (discussing *Melnick*).

Second, a physician might find a profusion greater than 1/0 but make a note that the disease is "not CWP, etiology unknown," as was the case in *Cranor*. *Id*. at 1-4. In that situation, the physician's comment is directed not to the presence of pneumoconiosis, but the etiology of the diagnosed pneumoconiosis. *Id*. at 1-5, 1-6. Accordingly, an administrative law judge should consider that type of comments later under 20 C.F.R. § 718.203 regarding the etiology of the claimant's pneumoconiosis.

Turning to Mr. K.'s claim, and with the *Cranor/Melnick* distinctions in mind, I note that on multiple occasions, Dr. Castle, Dr. Morgan, Dr. Fino, and Dr. Dahhan found the profusion category to be 1/1, but then indicated the opacities were not consistent with coal workers' pneumoconiosis. Since that type of interpretation is in the *Cranor* category, I will treat those chest interpretations as positive for pneumoconiosis and consider the etiology opinions under 20 C.F.R. § 718.203, if necessary.

Next, I also note that a substantial number of Mr. K.'s chest x-rays were obtained during treatment for various ailments. In many of those films, the physicians observed interstitial fibrosis but did not specifically identify pneumoconiosis or indicate the profusion and category of any opacities. For two reasons, I will treat those assessments as effectively negative for pneumoconiosis. First, since a physician evaluating a chest x-ray can be expected to accurately report the presence of any abnormalities, an administrative law judge may infer that the absence of a mention of pneumoconiosis indicates pneumoconiosis was not present. *See Marra v. Consolidation Coal Co.* 7 BLR 1-216, 1-219 (1985). Second, based on interpretations by several dual qualified radiologists who specifically noted the presence of interstitial fibrosis and also concluded the x-ray was negative for pneumoconiosis, I conclude that the interstitial fibrosis and pneumoconiosis are distinct radiographic findings.

Finally, while in two chest x-ray interpretations Dr. Smiddy only indicated the presence of interstitial fibrosis, I will nevertheless consider his December 1, 2003 and March 2, 2004 radiographic assessments positive for pneumoconiosis because he also correspondingly diagnosed coal workers' pneumoconiosis in his treatment notes and evaluations based in part on the two chest x-rays.

Date of x-ray	**Exhibit**	**Physician**	**Interpretation**
June 8, 1965	DX 2	Dr. Cunningham, B[13]	Negative for silicosis (pneumoconiosis).
Sept. 7, 1973	DX 1	Dr. R.H. Morgan, B	Negative for pneumoconiosis, pulmonary edema.

[13]The following designations apply: B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may posses to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs. *See also* 20 C.F.R. § 718.202(a)(1)(ii).

(same)	DX 1	Dr. Gale, B	Positive for pneumoconiosis, profusion category 1,[14] type t opacities,[15] mild interstitial fibrosis.
March 16, 1974	DX 2	Dr. Fino, B	Completely negative for pneumoconiosis, shows stable diffuse interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to coal workers' pneumoconiosis ("CWP"), most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	Negative for pneumoconiosis.
(same)	DX 1	Dr. J.P. Sutherland	Positive for pneumoconiosis, profusion category 2/3, type p opacities, emphysema present.
Jan. 29, 1980	DX 1 & DX 2	Dr. Hess	Positive for pneumoconiosis, profusion category 1/1, type p/s opacities.
Jan. 25, 1982	DX 2	Dr. Brandon	(Negative for pneumoconiosis) Calcified pulmonary parenchymal nodule consistent with old granulamatous disease.
Oct. 12, 1982	DX 2	Dr. Cunningham, B	(Negative for pneumoconiosis) Chronic changes in the lungs.
March 7, 1983	DX 2	Dr. Gale, B	Positive for pneumoconiosis, profusion category 2/2, type q/p opacities.
(same)	DX 2	Dr. Gaziano, B	Positive for pneumoconiosis, profusion category 1/1, type t opacities.
March 25, 1983	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	Negative for pneumoconiosis, insufficient profusion. Paraseptal emphysema and interstitial lung process present.

[14]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1/2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2.

[15]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

(same)	DX 2	Dr. Cunningham, B	Positive for pneumoconiosis, profusion category 2/1, type p/t opacities.
August 17, 1983	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	Negative for pneumoconiosis, only a few scant s/t opacities present.
(same)	DX 2	Dr. Cunningham, B	Positive for pneumoconiosis, profusion category 2/1, type p/t opacities.
August 20, 1983	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/0, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
(same)	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
May 5, 1986	DX 2	Dr. D. Shah	Positive for pneumoconiosis and chronic interstitial disease.
May 7, 1986	DX 2	Dr. D. Patel	(Negative for pneumoconiosis) COPD present.
May 19, 1986	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
Sept. 26, 1986	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
Nov. 14, 1986	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.

March 19, 1987	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
June 18, 1987	DX 2	Dr. Stephen Fisher, B, BCR	Positive for pneumoconiosis, profusion category 3/2, type s/p opacities, bullae and emphysema present.
(same)	DX 2	Dr. Marshall, B, BCR	Positive for pneumoconiosis, profusion category 3/2, type p/q opacities, emphysema present.
(same)	DX 2	Dr. Bassali, B	Positive for pneumoconiosis, profusion category 3/3, type s/t opacities, rule out (R/O) asbestosis.
June 3, 1988	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
April 12, 1989	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
Jan. 31, 1990	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
May 29, 1990	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.

(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
Oct. 1, 1991	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
Dec. 28, 1992	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
July 15, 1993	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
August 17, 1993	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
Jan. 31, 1994	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
May 2, 1994	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.

(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
March 1, 1995	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Dahhan	(Positive for pneumoconiosis), profusion 1/1, type s/t opacities; not consistent with coal workers' pneumoconiosis.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
March 30, 1995	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
April 3, 1995	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF (idiopathic pulmonary fibrosis).
(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, basilar changes indicate probable IPF.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to CWP, most likely bronchiectasis.
(same)	DX 2	Dr. Sridhar Iyengar,	(Negative for pneumoconiosis), accentuation of bronchovascular markings, patchy areas of infiltrative process.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.
May 30, 1995	DX 2	Dr. Fino, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities. Interstitial pattern in lung fields not consistent with occupational pneumoconiosis due to coal dust.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t opacities, not due to CWP, most consistent with bronchiectasis.
(same)	DX 2	Dr. W.K.C. Morgan, B	(Positive for pneumoconiosis), profusion category 1/1, type s/t opacities; characteristics inconsistent with CWP. Emphysema present.

Jan. 18, 1996	DX 2	Dr. D. Patel, BCR[16]	(Negative for pneumoconiosis), chronic interstitial changes in lungs.
March 28, 1996	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to CWP, most likely UIP or bronchiectasis.
Oct. 30, 1996	DX 2	Dr. William Lester	(Negative for pneumoconiosis), diffuse reticulonodular disease bilaterally, hyperexpansion of the lungs, large bullae
Jan. 5, 1997	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to CWP.
Jan. 8, 1997	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis), chronic changes in lungs.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to CWP (coal workers' pneumoconiosis), most likely UIP or bronchiectasis.
Feb. 27, 1997	DX 2	Dr. J.G. Patel	(Negative for pneumoconiosis), emphysematous chest.
April 30, 1997	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to CWP.
(same)	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis), chronic changes in lungs.
(same)	DX 2	Dr. Forehand, B	Positive for pneumoconiosis, profusion category 2/1, s/t opacities present, bi-basilar interstitial thickening, pleural changes present bilaterally.
July 21, 1997	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to CWP, most likely UIP or bronchiectasis.
April 19, 1998	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Pneumothorax on right side, increased bronchovascular markings, probably represent part of chronic changes or possible early infectious process.
(same)	DX 2	Dr. Weaver	(Negative for pneumoconiosis) Pneumothorax resolution, chronic changes.
April 20, 1998	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Mild pneumothorax, chronic changes.
April 21, 1998	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Emphysema present; otherwise "unremarkable."
April 23, 1998	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Emphysema present. Changes probably chronic with less likely possibility of CHF.
July 2, 1998	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.

[16]As I informed the parties at the hearing (TR, page 7), I take judicial notice of Dr. D. Patel's board certification and have attached the certification documentation.

(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. Castle, B	Positive for pneumoconiosis, profusion category 1/1, type t/u opacities, changes not due to CWP.
(same)	DX 2	Dr. John Weaver	No significant interval changes, chronic lung disease.
Sept. 18, 1998	DX 2	Dr. Hollenberg	(Negative for pneumoconiosis) Resolving large pneumothorax; mild interstitial fibrosis at lung bases.
Sept. 21, 1998	DX 2	Dr. Quillan	(Negative for pneumoconiosis) Moderate interstitial disease.
Sept. 25, 1998	DX 2	Dr. Fagan	(Negative for pneumoconiosis) Tiny, right pneumothorax; fibrotic changes present.
Sept. 28, 1998	DX 2	Dr. Burns	(Negative for pneumoconiosis) Small, right side pneumothorax; diffuse interstitial disease, most prominent at lung bases.
(same)	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, right pneumothorax, perhilar and basilar fibrosis consistent with IPF.
(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF (idiopathic pulmonary fibrosis).
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, fibrotic changes not due to CWP.
Oct. 2, 1998	DX 2	Dr. J. G. Patel	(Negative for pneumoconiosis) Pneumothorax present.
(same)	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis). Small pneumothorax, chronic changes in present.
Oct. 7, 1998	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, not due to changes of CWP
Oct. 16, 1998	DX 2	Dr. Castle, B	Positive for pneumoconiosis, profusion category 1/1, type t/u opacities; right chest tube, right pleural reaction, changes not due to CWP.
(same)	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Persistent chronic changes in lungs.
Oct. 17, 1998	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Persistent chronic changes in lungs.
Oct. 18, 1998	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis) No interval change in chest findings.
Oct. 19, 1998	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, no changes of CWP
(same)	DX 2	Dr. D. Patel, BCR	(Negative for pneumoconiosis), chronic changes with slight increased density right base.
Oct. 21, 1998	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, the changes are not due to CWP.
Oct. 27, 1998	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, pulmonary fibrosis at periphery and base of lungs.
(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, no changes due to CWP, right pleural thickening.
Feb. 2, 1999	DX 2	Dr. Spitz, B, BCR	Negative for pneumoconiosis, right pneumothorax, perhilar and basilar fibrosis consistent with IPF.

(same)	DX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, fibrotic changes at periphery of lung and lung bases consistent with IPF.
(same)	DX 2	Dr. Castle, B	(Positive for pneumoconiosis), profusion category 1/1, type t/u opacities, bullae and emphysema present. The changes are not due to CWP.
Oct. 10, 2001	DX 25	Dr. Wiot, BCR, B	Negative for pneumoconiosis. IPF present.
Oct. 17, 2001	DX 28	Dr. Ahmed, B, BCR	Positive for pneumoconiosis, profusion category 2/2, type t/u opacities, pleural thickening, emphysema, honeycombing and bullae present.
(same)	EX 2	Dr. Wiot, B, BCR	Negative for pneumoconiosis, severe bibasilar and midzone interstitial fibrosis, single nodular density in right interspace.
May 31, 2002	DX 17	Dr. Forehand, B	Positive for pneumoconiosis, profusion 3/2, type s/t opacities, pleural thickening.
(same)	DX 24, DX 30	Dr. Wheeler, B, BCR	Negative for pneumoconiosis, minimal to moderate linear interstitial fibrosis more likely than interstitial infiltrates in lower lungs; lateral periphery mid and upper lungs compatible with usual interstitial pneumonitis or collagen vascular disease.
June 26, 2002	CX 5	Dr. Alexander, B, BCR	Positive for pneumoconiosis, profusion 3/2, type t/s opacities, some q and r opacities are present in the right middle and upper zones, bilateral pleural thickening, enlarged heart.
(same)	DX 23	Dr. Castle, B	Negative for pneumoconiosis; evidence of extensive bilateral interstitial fibrosis consistent with idiopathic pulmonary fibrosis.
January 30, 2003	CX 1	Dr. Ahmed, B, BCR	Positive for pneumoconiosis, profusion 2/1, type t/u opacities, pleural thickening, emphysema, and bullae present.
(same)	CX 4	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Significant extensive chronic interstitial changes, emphysema
(same)	EX 9	Dr. Scatarige, B, BCR	Negative for pneumoconiosis, moderate bilateral interstitial fibrosis, few bullae present in right upper lobe.
September 10, 2003	CX 4	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Moderately hyperinflated lungs with extensive interstitial markings indicates underlying chronic change; suggestion of superimposed interstitial edema.
September 15, 2003	CX 4	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Chronic interstitial changes in the lungs; interstitial lung pattern is due to interstitial fibrosis rather than interstitial edema.
October 17, 2003	CX 4	Dr. Patrick Rao, BCR[17]	(Negative for pneumoconiosis) Advanced bullous emphysema; no acute lung abnormality noted.
December 1, 2003	CX 4	Dr. Smiddy	(Positive for pneumoconiosis) Cardiomegaly, five lobe interstitial fibrosis, minimal old granulomas, bilateral pleural thickening.
February 16, 2004	CX 4	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Prominence of bronchovascular markings and interstitial lungs markings with patchy infiltrates in the left lower lung field, suggestive of acute process like pneumonia.

[17]I take judicial notice of Dr. Rao's board certification and have attached the certification documentation.

February 19, 2004	CX 4	Dr. D. Patel, BCR	(Negative for pneumoconiosis) Chronic changes with interval development of pneumonia and a small left sided pleural effusion.
March 2, 2004	CX 4	Dr. Smiddy	(Positive for pneumoconiosis) Cardiomegaly, five lobe interstitial fibrosis; no change from December 2003 film.
April 20, 2004	EX 4	Dr. Wheeler, B, BCR	Negative for pneumoconiosis, borderline cardiomegaly, moderate linear interstitial fibrosis more likely than interstitial infiltrates in lower lungs.

Based on the undisputed interpretations, the following 24 films are negative for pneumoconiosis: June 8, 1965, January 25, 1982, October 12, 1982, May 7, 1986, January 18, 1996, October 30, 1996, February 27, 1997, April 19, 1998, April 20, 1998, April 21, 1998, April 23, 1998, September 18, 1998, September 21, 1998, September 25, 1998, October 2, 1998, October 17, 1998, October 18, 1998, October 10, 2001, September 10, 2003, September 15, 2003, October 17, 2003, February 16, 2004, February 19, 2004, and April 20, 2004.

Likewise, in the absence of any contrary assessment, the following 29 films that are positive for pneumoconiosis: January 29, 1980, March 7, 1983, August 20, 1983, May 5, 1986, May 19, 1986, September 26, 1986, November 14, 1986, March 19, 1987, June 18, 1987, June 3, 1988, April 12, 1989, January 31, 1990, May 29, 1990, October 1, 1991, December 28, 1992, July 15, 1993, August 17, 1993, January 31, 1994, May 2, 1994, March 1, 1995, March 30, 1995, May 30, 1995, March 28, 1996, January 5, 1997, July 21, 1997, October 7, 1998, October 21, 1998, December 1, 2003, and March 2, 2004.

Based on the conflicting interpretations, the following 5 chest x-rays are inconclusive on the presence of pneumoconiosis: September 7, 1973 (Morgan v. Gale, both B readers), October 16, 1998 (Dr. Patel, board certified radiologist v. Dr. Castle, B reader), October 19, 1998 (Dr. Patel v. Dr. Castle), October 17, 2001 (Ahmed v. Wiot, both dual qualified radiologists), and January 30, 2003 (Ahmed v. Scatarige, both dual qualified radiologists).

In the March 16, 1974 x-ray, Dr. Fino and Dr. Morgan, B-readers, did not observe the presence of pneumoconiosis. However, Dr. Castle, a B-reader, and Dr. Sutherland believed the x-ray was positive for pneumoconiosis. Since the interpretations by two B-readers outweigh the contrary opinions by one B-reader and another physician, the x-ray is negative for pneumoconiosis

In the March 25, 1983 chest x-ray, Dr. Castle, Dr. Fino, and Dr. Cunningham observed the presence of pneumoconiosis; Dr. Morgan did not. Since all four physicians are B readers, the consensus of Dr. Castle, Dr. Fino, and Dr. Cunningham establishes that that March 25, 1983 film is positive for pneumoconiosis.

In the August 17, 1983 chest x-ray, Dr. Castle and Dr. Cunningham observed the presence of pneumoconiosis; Dr. Morgan did not. Since all three physicians are B readers, the consensus of Dr. Castle and Dr. Cunningham establishes that that August 17, 1983 film is positive for pneumoconiosis.

Concerning the April 3, 1995 film, Dr. Spitz and Dr. Wiot, both dual qualified radiologists, and Dr. Iyengar did not observe the presence of pneumoconiosis. On the other hand, Dr. Castle, Dr. Fino, and Dr. Morgan, B-readers, believed pneumoconiosis was present in the x-ray. The consensus by two dual qualified radiologists outweighs the contrary opinions by physicians not similarly qualified.[18] The April 3, 1995 film is therefore negative for pneumoconiosis.

A similar medical conflict exists in the January 8, 1997 film. Dr. Spitz and Dr. Wiot, along with Dr. Patel, a board certified radiologist, believed the x-ray to be negative for pneumoconiosis. Dr. Castle, a B-reader, observed opacities consistent with pneumoconiosis. Once again, the preponderance of the interpretations by the better qualified physicians establishes the January 8, 1997 film is negative.

The same physicians came to the same conclusions with regard to the April 30, 1997 film with the addition of Dr. Forehand, a B-reader, finding the presence of pneumoconiosis. Nonetheless, the negative interpretations by Dr. Spitz and Dr. Wiot, the better qualified radiologists, outweigh the contrary opinion by Dr. Castle and Dr. Forehand and that film is negative as well.

Similarly, concerning the interpretation conflicts with the chest x-rays of July 2, 1998, September 28, 1998, October 27, 1998, and February 2, 1999, the negative interpretations by the best qualified physicians, Dr. Wiot and Dr. Spitz, outweigh the opinions by the other less qualified physicians, establishing the four films are negative for pneumoconiosis.

Dr. Wheeler, a dual qualified radiologist, interpreted the May 31, 2002 film as negative for pneumoconiosis. Dr. Forehand, a B-reader, observed the presence of pneumoconiosis. Due to Dr. Wheeler's better credentials, the May 31, 2002 film is negative.

Finally, in the June 26, 2002 x-ray, Dr. Alexander, the better qualified radiologist, found the presence of pneumoconiosis; Dr. Castle did not. This film is therefore positive.

In summary, 33 chest x-rays are negative for the presence of pneumoconiosis; 32 are positive and 5 are inconclusive. Consequently, since the preponderance of the chest x-ray evidence is essentially inconclusive, Mr. K. is unable to establish the presence of pneumoconiosis in his lungs by radiographic evidence under 20 C.F.R. § 718.202(a)(1).

Additional Discussion

The chest x-ray evidence is effectively inconclusive for the presence of pneumoconiosis. However, one additional consideration demonstrates that the preponderance of the radiographic

[18]*See Zeigler Coal Co. v. Director [Hawker]*, 326 F.3d 894 (7th Cir. 2003); *Cranor v. Peabody Coal Co.*, 22 B.L.R. 1-1 (1999) (en banc on recon.) (greater probative weight may be given to the interpretations of a dual qualified radiologist in comparison to a physician who is only a B reader.)

evidence weighing against a finding of clinical coal workers' pneumoconiosis is much greater than the above summary demonstrates. Of the 32 positive for pneumoconiosis films, 25 of the positive findings were significantly based on the interpretations by Dr. Castle and Dr. Morgan, who specifically indicated that the pneumoconiosis they observed in the films was not consistent with coal workers' pneumoconiosis. The physicians explained that the irregular shape of the pneumoconiosis opacities, their location in the lung bases, and the absence of rounded opacities indicated they were not caused by coal mine dust. Dr. Morgan further explained that coal workers' pneumoconiosis first develops in the upper and mid lung zones as rounded opacities before irregular opacities related to coal mine dust appear in the lower lobes. During his review of the radiographic record, Dr. Rosenberg also noted that coal workers' pneumoconiosis is characterized by upper lobe predominance of micronodules, which Mr. K.'s chest x-rays do not contain. Thus, even if positive interpretations had represented the preponderance of radiographic evidence, establishing clinical pneumoconiosis under 20 C.F.R. § 718.202(a)(1), Mr. K. would have difficulty establishing the cause of the pneumoconiosis. Based on his length of coal mine employment, a presumption exists under 20 C.F.R. § 718.203 that any pneumoconiosis in his lungs is related to his coal mine employment. However, that presumption is rebuttable and the notations by Dr. Castle, Dr. Fino, and Dr. Morgan, coupled with Dr. Morgan's assessment represents sufficient evidence to rebut that presumption.

Medical Opinion

Although Mr. K. cannot establish the presence of black lung disease through the preponderance of chest x-ray evidence, he may still prove this requisite element of entitlement under 20 C.F.R. § 718.202(a)(4) through the preponderance of the more probative medical opinion. Prior to considering the various medical assessments of Mr. K.'s pulmonary condition, a review of the other medical evidence in the record helps to understand the medical opinions.

Chest CT Scan – October 17, 2003
(CX 4 and EX 8)

On October 17, 2003, Dr. Patrick Rao, a board certified radiologist, conducted a CT scan of Mr. K.'s chest. He found diffuse emphysema as well as chronic bronchitis. "Mediastinal windows demonstrate scattered vascular calcifications." Dr. Rao noted "no definite air space disease or pulmonary nodules."

Dr. Jerome Wiot, a B-reader and board certified radiologist, also reviewed the October 17, 2003 x-ray and found no evidence of coal workers' pneumoconiosis. The physician observed extensive bullous change in the right upper lung field with less significant bullous change throughout with emphysema. There is also basilar interstitial disease, which is not a manifestation of coal dust exposure. Coal workers' pneumoconiosis starts in the upper lung fields and only when the disease progresses, it moves to the mid and lower lung fields. The findings are therefore not consistent with coal workers' pneumoconiosis.

Pulmonary Function Tests

Exhibit	Date / Doctor	Age / Height	FEV¹ pre[19] post[20]	FVC pre post	MVV pre post	% FEV¹ / FVC pre post	Qualified[21] pre Post	Comments
DX 1	Nov. 5, 1979 Dr. Hatfield	50 67"	3.05	4.2	142	72.6%	No[22]	(Invalid.)
DX 2	Mar. 25, 1983 Dr. Abernathy	53 65"	2.39	3.11	70	77%	No[23]	Small airways obstruction.
DX 2	Aug. 17, 1983 Dr. Abernathy	54 66"	2.36 2.40	3.08 3.08	76 80	77% 76%	No[24]	
DX 2	March 1, 1995 Dr. Dahhan	65 65.5"	2.03 1.96	3.38 3.12	61 58	60.0% 62.8%	No[25]	Mild obstructive / restrictive defect, severe diffusion defect.
DX 2	July 2,1998 Dr. Thakkar	69 66"	2.01 2.02	2.94 2.79	---	68% 72%	No[26]	
DX 2	Feb. 2, 1999 Dr. Castle	69 64″	2.12 2.26	2.98 3.14	78 73	71.1% 72.0%	No[27]	Normal, diffusion moderately reduced.

[19]Test result before administration of a bronchodilator.

[20]Test result following administration of a bronchodilator.

[21]Under 20 C.F.R. § 718.204 (b)(2)(i), to qualify for total disability based on pulmonary function tests, for a miner's age and height, the FEV1 must be equal to or less than the value in Appendix B, Table B1 of 20 C.F.R. § 718, **and either** the FVC has to be equal or less than the value in Table B3, or the MVV has to be equal **or** less than the value in Table B5, or the ratio FEV1/FVC has to be equal to or less than 55%.

[22]The qualifying FEV1 number is 1.97 for age 50 and 67″; the corresponding qualifying FVC and MVV values are 2.48 and 79, respectively.

[23]The qualifying FEV1 number is 1.77 for 65" and age 53.

[24]The qualifying FEV1 number is 1.81 for 66" and age 54.

[25] The qualifying FEV1 number is 1.60 for age 65 and 65.4″; the corresponding qualifying FVC and MVV values are 2.07 and 64, respectively.

[26]The qualifying FEV1 number is 1.57 for 66" and age 69.

[27]The qualifying FEV1 number is 1.41 for age 69 and 63.8″; the corresponding qualifying FVC and MVV values are 1.84 and 55, respectively.

DX 8	May 31, 2002 Dr. Forehand	73 64”	2.04	2.91	61	70.1%	No[28]	
DX 23	June 26, 2002 Dr. Castle	73 65”	1.82 1.96	2.55 2.84	41	71.4% 69.0%	No[29] No	No large airway obstruction, mild restriction, diffusion is severely reduced.
DX 28	Jan. 15, 2003 Dr. Narayanan	73[30] 66”	1.68	2.39		70.3%	No[31]	Moderate to severe airways obstruction[32]
EX 4	April 20, 2004 Dr. Rosenberg	74 65”	1.44	2.01	38	71.6	Yes	

Additional Arterial Blood Gas Studies

Exhibit	**Date / Doctor**	**pCO_2 (rest) pCO_2 (exercise)**	**pO_2 (rest) pO_2 (exercise)**	**Qualified[33]**	**Comments**
DX 1	Nov. 23, 1979 Dr. Hatfield	33.9 13.2	71 84	No[34] No[35]	
DX 2	Jan. 25, 1982 Dr. Marsi	35	72	No[36]	
DX 2	Jan. 27, 1982 Dr. Marsi	36	73	No[37]	

[28]The qualifying FEV1 number is 1.38 for age 71 and 63.8″; the corresponding qualifying FVC and MVV values are 1.81 and 55, respectively.

[29]The qualifying FEV1 number is 1.48 for age 71 and 65″; the corresponding qualifying FVC and MVV values are 1.92 and 59, respectively.

[30]The test incorrectly listed Mr. K.’s age as 63.

[31]The qualifying FEV1 number is 1.54 for age 71 and 65.7″; the corresponding qualifying FVC and MVV values are 2.00 and 62, respectively.

[32] Dr. Renn, board certified in pulmonary diseases and internal medicine, believes the spirometry study is invalid for accurate interpretation or for the derivation of significant data to assess Mr. K.’s ventilatory function (EX 1).

[33]To qualify for Federal Black Lung Disability benefits at a coal miner’s given pCO_2 level, the value of the coal miner’s pO_2 must be equal to or less than corresponding pO^2 value listed in the Blood Gas Tables in Appendix C for 20 C.F.R. § 718.

[34]For the pCO_2 of 33, the qualifying pO_2 is 67 or less.

[35]For the pCO_2 of 25 or below, the qualifying pO_2 is 75 or less.

[36]For the pCO_2 of 35, the qualifying pO_2 is 65 or less.

[37]For the pCO_2 of 36, the qualifying pO_2 is 64 or less.

DX 2	Feb. 17, 1982 Dr. Thakkar	40 20	73 134	No[38] No	
DX 2	March 25, 1983 Dr. Abernathy	36 32	61 68	Yes Yes[39]	
DX 2	Aug. 17, 1983 Dr. Abernathy	35	64	Yes	
DX 2	May 5, 1986 Dr. Sutherland	39	65	No[40]	
DX 2	March 1, 1995 Dr. Dahhan	34.4 34.5	63.5 64	Yes[41] Yes	
DX 2	Feb. 2, 1999 Dr. Castle	36.3	68.5	No	
DX 2	July 2, 1998 Dr. Thakkar	34.6	65.4	Yes	
DX 15	May 31, 2002 Dr. Forehand	32 29	58 39	Yes Yes[42]	
DX 28, CX 2, CX 4	Feb. 28, 2002 Dr. Thakkar	32	50.7	Yes	
DX 23	June 26, 2002 Dr. Castle	34.3	50.2	Yes	Resting ABG shows severe hypoxemia.
EX 4	April 20, 2004 Dr. Rosenberg	34.6	39.7	Yes	Markedly reduced oxygenation.

Dr. John R. Hatfield
(DX 1)

On November 23, 1979, Dr. Hatfield conducted a pulmonary evaluation of Mr. K. who was working as a coal miner and had smoked a pack of cigarettes a day since he was 20 years old. Mr. K. complained about chronic shortness of breath and cough. Upon physical examination, Dr. Hatflied heard bilateral wheezes. The pulmonary function test showed a severe obstruction but was of questionable validity. A chest x-ray was not authorized. Dr. Hatfield concluded Mr. K. had possible pneumoconiosis and severe obstructive disease.

Dr. Ralph W. Hess
(DX 1)

On February 5, 1980, Dr. Hess noted that Mr. K. had 32 years of coal mine employment and suffered from shortness of breath with exercise. Mr. K. had also suffered a lumbosacral sprain in a 1980 mining accident. A chest x-ray was positive for pneumoconiosis.

[38]For the pCO_2 of 40 to 49, the qualifying pO_2 is 60 or less.

[39]For the pCO_2 of 32, the qualifying pO_2 is 68 or less.

[40]For the pCO_2 of 39, the qualifying pO_2 is 61 or less.

[41]For the pCO_2 of 34, the qualifying pO_2 is 66 or less.

[42]For the pCO_2 of 29, the qualifying pO_2 is 71 or less.

Dr. F. A. Masri
(DX 2)

For nine days at the end of January 1982, Dr. Masri treated Mr. K. in the hospital for shortness of breath and emphysematous lungs. On physical examination, Mr. K. had prolonged breath sounds. The chest x-ray showed old granulomatous disease. Dr. Masri diagnosed acute exacerbation of asthmatic bronchitis and COPD.

In October 1982, Dr. Masri hospitalized Mr. K. for a respiratory infection. The physician diagnosed COPD.

Dr. Robert Abernathy
(DX 2)

On April 5, 1983 and March 16, 1984, Dr. Abernathy, board certified in internal medicine, conducted pulmonary evaluations of Mr. K, who mined coal for 35 years and was presently smoking about half a pack of cigarettes a day. He reported significant shortness of breath and wheezing. Upon physical examination, the chest sounds were clear. Two chest x-rays were positive for pneumoconiosis and the arterial blood gas revealed an oxygenation problem. Dr. Abernathy determined that Mr. K.'s arterial oxygen level had decreased significantly and that cigarette smoking had a greater influence on Mr. K.'s pulmonary function than "the exposure to coal workers' pneumoconiosis." The degree of coal workers' pneumoconiosis in the chest x-ray was not associated with the type of Mr. K.'s pulmonary function impairment. Since his oxygen tension increased with exercise, the test result was more consistent with chronic bronchitis. Coal workers' pneumoconiosis is also not the cause of Mr. K.'s impairment because he has an obstructive component of airflow through small airways which is consistent with an impairment due to cigarette smoking. Dr. Abernathy concluded that Mr. K. has coal workers' pneumoconiosis but any impairment in pulmonary function that he has comes from smoking. Therefore, any disability is related to inhalation of smoke, not coal dust.

Dr. J.P. Sutherland
(DX 2)

Between May 5 and 9, 1986, Dr. Sutherland hospitalized Mr. K. for acute shortness of breath, exacerbation of COPD, severe pneumoconiosis, acute bronchitis, and pneumonia. Upon physical examination, Dr. Sutherland heard diffuse rhonchi. Chest x-rays indicated the presence of pneumoconiosis.

Dr. W.K.C. Morgan
(DX 2)

On February 11, 1988, Dr. Morgan conducted a medical record review in Mr. K.'s case. He found a mild restrictive impairment, sometimes mild hypoxemia, and although an x-ray shows the presence of opacities, they are unusual for coal workers' pneumoconiosis. Persistent changes over time in Mr. K.'s lungs indicate interstitial fibrosis. The x-ray is abnormal but Dr.

Morgan was uncertain of the etiology. It "seem[s]" to be consistent with interstitial fibrosis, not coal workers' pneumoconiosis.

On February 27, 1988, in a supplemental report, Dr. Morgan noted that Mr. K.'s minor abnormalities of lung function indicate the presence of respiratory impairment and abnormal gas exchange. These symptoms are not compatible with coal workers' pneumoconiosis and more consistent with interstitial fibrosis unrelated to Mr. K.'s coal mine employment. Mr. K. does not have coal workers' pneumoconiosis.

On October 11, 1995, Dr. Morgan reviewed additional pulmonary evaluations and treatment records. Dr. Morgan observed that Mr. K developed significant hypoxemia and some restrictive impairment associated with obesity or interstitial fibrosis. Concerning the chest x-rays, Dr. Morgan did not believe that Mr. K. has coal workers' pneumoconiosis. His chest x-rays show irregular opacities in the lower lobes, "which is not the site where CWP begins and in addition CWP is characterized by round opacities first appearing in the upper zones." In his opinion, the irregular opacities in the lower lobes, coupled with the restrictive impairment and a reduction of diffusion capacity support a diagnosis of interstitial fibrosis, sometimes called "fibrosing alveolitis." This disease occurs in old persons due to an unknown cause. Dr. Morgan concluded Mr. K. is totally disabled due to heart disease and possible interstitial fibrosis.

After reviewing a number of chest x-rays taken of Mr. K. over time, on November 2, 1995, Dr. Morgan noted that the appearance of the chest x-rays are not compatible with coal workers' pneumoconiosis but are compatible with an airflow limitation due to emphysema and smoking. Specifically, although the profusion of the opacities was sufficient for a 1/1 determination, the characteristics of the opacities and their irregular shape of the opacities, the location of irregular opacities solely in the lower lobes, and the notable absence of rounded opacities in the middle and upper lung zones, indicated the radiographic opacities are not coal workers' pneumoconiosis or related to coal mine dust exposure. Dr. Morgan also observed centrilobular emphysema. Dr. Morgan believed that Mr. K.'s lung function abnormalities consisted of a restrictive defect and a reduction in diffusion capacity related to interstitial fibrosis. The emphysema and the airway obstruction could be due to cigarette smoking or the interstitial fibrosis. The later diagnosis is also compatible with the pulmonary function tests. Mr. K. is impaired to some extent, making heavy labor difficult. His impairment is not related to coal dust or coal workers' pneumoconiosis but to cigarette smoke, even if coal workers' pneumoconiosis were present. Although a CT scan might be helpful, Dr. Morgan anticipated that it would not show the presence of coal workers' pneumoconiosis.

Dr. Gregory J. Fino
(DX 2)

On February 24, 1988, Dr. Fino, board certified in pulmonary disease and internal medicine, conducted a pulmonary medical record review. Mr. K. had mined coal for 35 years and was a long term cigarette smoker. The pulmonary function tests produced no evidence of significant lung fibrosis because diffusing capacity was normal. Mr. K. has a restriction and mild hypoxia may be due to cigarette smoking and coal workers' pneumoconiosis. However, the hypoxia improves with exercise, which indicates that it is not related to coal workers'

pneumoconiosis. Mr. K. has a mild respiratory impairment but could return to his previous coal mining job. He is, however, disabled as a whole person due to a cardiac condition. Dr. Fino concluded that Mr. K. has simple coal workers' pneumoconiosis and a mild respiratory impairment which was not due to coal workers' pneumoconiosis.

On March 1, 1988, upon review of Mr. K.'s hospitalization records, Dr. Fino noted the attending physicians reported wheezing which is indicative of asthmatic bronchitis secondary to cigarette smoking. Although industrial bronchitis may occur during employment, it subsides after a person is removed from exposure. As a result, Dr. Fino does not attribute Mr. K.'s asthmatic bronchitis to his exposure to coal mine dust.

On April 26, 1995, Dr. Fino reviewed Mr. K.'s medical records. He found that coal workers' pneumoconiosis was present and that his diffusion capacity worsened between 1983 and 1995. The pattern of opacities suggests the obstruction is secondary to cigarette smoking. Mr. K. retains the capacity to perform his previous coal mining job. He is not totally disabled and any lung impairment related to obstruction that he has results from smoking.

On March 1, 1998, Dr. Fino conducted a supplemental medical record review. Mr. K. had been admitted to the hospital multiple times for wheezing and acute exacerbation of underlying obstructive lung disease; however, upon evaluation, Dr. Fino did not diagnose Mr. K. with an obstruction. Dr. Fino opined that Mr. K. suffered from asthmatic bronchitis secondary to smoking. His condition improved with medications. Pneumoconiosis, however, causes a fixed impairment. Although a coal miner can be diagnosed with asthmatic bronchitis, it usually occurs while the miner is actually working in the mines and the other evidence, including no obstruction and a mild restriction, suggests that the condition is not the result of coal dust exposure.

Dr. J. G. Patel
(DX 2)

On October 28, 1994, Dr. Patel evaluated Mr. K., who presented with progressive shortness of breath, productive cough, and a history of angina pectoris and cardiac catherization. He worked in the coal mines for 35 years and smoked 1 pack of cigarettes per day for 20 years. Mr. K. has mild respiratory distress. A physical exam revealed significantly reduced breath sounds in both lung fields with scattered rhonchi. The pulmonary function test showed a mild to moderate obstructive airways disease with fairly good response to bronchodilation. The x-ray had increased bronchovascular markings suggestive of COPD and coal workers' pneumoconiosis. Dr. Patel concluded that Mr. K. has significant underlying lung disease that is causing significant disability and precludes Mr. K. from returning to his previous coal mine employment.

On January 12, 1995, Dr. Patel treated Mr. K., who had a history of COPD with recurrent bronchitis, shortness of breath, especially on exertion and productive cough. A physical exam of the chest revealed reduced breath sounds toward the base. Dr. Patel included "possible" coal workers' pneumoconiosis in his diagnosis.

On February 27, 1997, Dr. Patel noted that Mr. K. had an emphysematous chest.

On April 20, 1998, Dr. Patel again treated Mr. K., noting his medical history, which included underlying coal workers' pneumoconiosis and heart disease. Mr. K. reported smoking one pack of cigarettes per day for 20 years and working in the coal mines for 35 years.

Dr. Patel treated Mr. K. for a pneumothorax in September/October 1998. He noted chronic changes in the lungs with slight increased density in the right base.

Dr. Abdul Dahhan
(DX 2)

On March 1, 1995, Dr. Dahhan, board certified in pulmonary and internal medicine, conducted a pulmonary evaluation of Mr. K.. He also testified in a deposition on November 9, 1995 regarding his findings. Mr. K. complained of daily cough, sputum production, wheezing, shortness of breath, chest pain, and dyspnea. His medical history includes an empyema, for which surgery was conducted on his left lung when he was 10 years old. Mr. K. reports a cigarette smoking history of 36 pack-years[43]. He worked in the coal mines for 35 years, ending his coal mine employment as a section foreman. The x-ray showed opacities but not the type or location of opacities consistent with coal workers' pneumoconiosis. The ventilatory study showed a mild obstructive defect and the lung volume study revealed a mild restrictive defect. These abnormalities along with the shape and location of the opacities are consistent with interstitial lung disease.

In Dr. Dahhan's opinion, Mr. K. has moderate respiratory impairment attributable to smoking. His mild obstructive defect is not reversible and he has moderate hypoxia. Mr. K. does not retain the capacity to return to coal mine employment given the heavy labor job that he described. Although the x-ray fits with the pneumoconiosis classification, it is not consistent with coal workers' pneumoconiosis. Mr. K. has a respiratory impairment but it is not caused by coal dust exposure. Rather, his smoking history has caused airway obstruction and coronary artery disease. Even if Mr. K. were found to have coal workers' pneumoconiosis, it does not contribute to his total disability, which was caused by smoking.

Dr. D. Scott Andrews
(DX 2)

From September 18 through September 28, 1998, Dr. Andrews hospitalized Mr. K. for spontaneous pneumothorax and severe COPD. During the course of treatment, chest x-rays showed severe pneumothorax. Mr. K.'s COPD precluded surgery so he was discharged with a chest tube and valve.

[43]A pack-year equals the consumption of one pack of cigarettes a day for one year.

Dr. Charles H. Edwards
(DX 2)

On December 1, 1998, Dr. Edwards indicated that Mr. K.'s COPD was caused by both pulmonary risk factors; chronic cigarette smoking and exposure to coal mine dust.

Dr. Joseph Smiddy
(CX 3 and CX 4)

On October 15, 2003, Dr. Smiddy, board certified in internal medicine, conducted a pulmonary evaluation of Mr. K. upon referral. Dr. Smiddy noted that Mr. K. has profound hypoxia, cyanosis, oxygen dependency, white sputum, orthopnea, and dyspnea on exertion. With oxygen, Mr. K. can only walk 100 feet without stopping. He has a history of chest pain and coronary artery disease. He has underlying COPD, coal workers' pneumoconiosis, and heart disease. Mr. K. worked in coal mine employment for 35 years loading coal, working on a cutting machine and working on a continuous miner as section foreman. He has had pneumonia and hypertension in the past. He smoked in the past but quit 14 years ago. Prior chest x-rays revealed extensive chronic interstitial changes which apparently have been present for a long time.

A physical exam of the chest showed that it was consistent with age and status. Prior chest x-rays revealed that Mr. K. has coal workers' pneumoconiosis with borderline heart enlargement. He also has diffuse interstitial prominence of markings and "a broad different diagnosis would be possible and multiple concomitant diagnoses would be possible." Dr. Smiddy diagnosed interstitial lung disease, COPD, coal workers' pneumoconiosis, coronary artery disease, and possible element of congestive heart failure in addition to sleep apnea, deconditioning, and hypoxia.

On October 22, 2003, Dr. Smiddy followed up on Mr. K.'s pulmonary condition. He noted Mr. K. has COPD, interstitial lung disease and significant emphysema with coal workers' pneumoconiosis. Physical exam of the chest produced normal results. Dr. Smiddy diagnosed the same conditions as in his prior visit with the patient.

On December 1, 2003, Dr. Smiddy treated Mr. K. for increased shortness of breath, wheeze and cough. His condition was unchanged since a previous exam on October 15, 2003.

On January 12, 2004, Dr. Smiddy evaluated Mr. K.'s pulmonary condition. Dr. Smiddy noted Mr. K.'s known COPD, coal workers' pneumoconiosis and interstitial lung disease. Mr. K. presented with chronic shortness of breath, cough, and wheeze. A chest exam revealed symmetrical percussion and palpitation, and auscultation was clear without rales, rubs, or rhonchi. Dr. Smiddy diagnosed COPD, coal workers' pneumoconiosis, and interstitial lung disease.

On March 2, 2004, Dr. Smiddy evaluated Mr. K.'s pulmonary condition. Mr. K. has interstitial lung disease and underlying coal workers' pneumoconiosis and COPD. A chest x-ray revealed cardiomegaly and five lobe interstitial fibrosis.

On April 13, 2004, Dr. Smiddy evaluated Mr. K.'s pulmonary condition. Dr. Smiddy noted that Mr. K. has interstitial lung disease, COPD, and coal workers' pneumoconiosis and is extremely short of breath and cyanotic at rest on four liters of oxygen with a SAT of 78% walking and 87% at best. Mr. K. declined a biopsy because he felt he was too ill to have one.

Dr. Muljibhai Thakkar[44]
(DX 2, DX 28, and CX 4)

On October 18, 1994, Dr. Thakkar, board certified in internal medicine and cardiology and Mr. K.'s treating physician since December 1981, summarized his treatment of Mr. K. Mr. K. initially presented with a history of COPD secondary to probable coal workers' pneumoconiosis. He complained of shortness of breath with 4 to 5 steps. He sleeps with 2 pillows. A physical exam of the chest revealed moderate emphysema, and hyper-resonance. Mr. K. has a moderately restrictive lung disease with a slightly superimposed obstructive disease. Mr. K.'s x-rays show COPD with chronic interstitial changes. Dr. Thakkar diagnosed COPD secondary to coal workers' pneumoconiosis and moderate obstructive disease. Mr. K.'s lung impairment is caused by rock dust from coal mine employment for 35 years.

On July 2, 1998, Dr. Thakkar reported that pulmonary function tests showed Mr. K.'s FVC was mildly reduced and found no significant obstruction.

On September 4, 1998, Dr. Thakkar reported Mr. K.'s history of COPD secondary to coal workers' pneumoconiosis, angina pectoris, and cardiac catherization. Mr. K. complained of dyspnea on exertion. Mr. K. has been hospitalized several times for pneumonia, high grade fever, and cough. The physician noted a long period of chest x-rays showing chronic interstitial changes and diffuse reticulonodular disease. The physician concluded that Mr. K.'s coal workers' pneumoconiosis which caused his lung problem to become severe.

Between January 1997 and May 1999, Dr. Thakkar treated Mr. K. for various illnesses and complaints. On one occasion when Mr. K. had pneumonia, Dr. Thakkar heard rales in the lung bases. At other times, physical examination disclosed emphysematous chest.

On November 13, 2000, Dr. Thakkar stated that Mr. K. was totally disabled in part due to coal workers' pneumoconiosis. Mr. K. worked over 30 years in the coal mines. In his last job as a section foreman, Mr. K. engaged in constant walking and strenuous tasks, working with the men under his supervision. Based on pulmonary function test results, Dr. Thakkar concludes Mr. K. is no longer capable of walking long distance or other coal mining activities. Mr. K. had a "confirmed" diagnosis of pneumoconiosis. His cigarette smoking covered 18 to 36 pack-years and is comparable to his coal mine dust exposure. Consequently, Dr. Thakkar believes both exposures contributed "in similar amounts to his disabling respiratory impairment." He also struggles with COPD, angina, and episodic congestive heart failure. The COPD symptoms include chronic shortness of breath, decreasing lung function, and susceptibility to other pulmonary disease.

[44]I have not included notes from Dr. Thakkar's treatment of non-pulmonary ailments.

On April 9, 2002, Dr. Thakkar summarized his medical opinion about Mr. K. Dr. Thakkar diagnosed Mr. K. with a chronic respiratory or pulmonary condition due to coal dust exposure in whole or in part. Mr. K. worked for 30 years in coal mine employment as section foreman, which required constant walking and other strenuous tasks. Chest x-rays showed chronic interstitial changes consistent with COPD, chronic lung disease, and emphysema. Mr. K. complained of shortness of breath, cough, ASHD, hypertension, chronic stable angina, and COPD. Dr. Thakkar prescribed supplemental oxygen therapy. The severity of Mr. K.'s lung problems contributes to his heart problems. The pulmonary function test shows Mr. K. has a breathing impairment and can no longer perform his last coal mining job, rendering him totally disabled. Mr. K. has coal workers' pneumoconiosis "due to occupational dust exposure for 33 years."

On September 10, 2003, Dr. Thakkar examined Mr. K. in the hospital. He presented with cough, chest congestion and shortness of breath for four days. Dr. Thakkar reported Mr. K.'s known COPD secondary to coal workers' pneumoconiosis, heart disease, status post angioplasty, and status post coronary artery disease. Mr. K. also had a stroke in the past. Mr. K. was referred to the emergency room after undergoing a black lung examination where it was discovered that he was in moderate respiratory distress. Mr. K. smoked one pack of cigarettes per day for about 20 years, quitting 13 years ago.

A physical examination showed Mr. K.'s chest to be moderately emphysematous. His lungs were hyperresonant on percussion and air entry was moderately diminished throughout the chest with few scattered rales and rhonchi at the right base. Dr. Thakkar believed Mr. K. had acute bronchitis versus pneumonia and acute exacerbation of COPD. Secondary diagnoses included arteriosclerotic heart disease and COPD secondary due to CWP. Mr. K. was admitted to the hospital.

On September 11, 2003, Dr. Thakkar performed an echocardiogram on Mr. K. The physician found borderline left atrial size, borderline left ventricular size, thickened aortic leaflets, mildly enlarged right atrium and right ventricular cavity, and mild tricuspid valve insufficiency.

Mr. K. was discharged from the hospital on September 17, 2003. In the discharge summary, his progress throughout his seven-day stay was noted. A chest x-ray taken on September 10, 2003 showed a suggestion of superimposed interstitial edema and a repeat chest x-ray showed chronic changes in the lungs probably due to interstitial fibrosis rather than interstitial edema. By September 17, Mr. K.'s condition stabilized and he was released from the hospital. His discharge diagnosis included acute bronchitis that had been controlled, acute exacerbation of COPD that was under control, and improved congestive heart failure. His secondary diagnoses remained the same.

On February 16, 2004, Mr. K. was admitted to the hospital after coughing, smothering, and experiencing fever and chills for three days with pneumonia and shortness of breath. A chest exam revealed a moderately emphysematous chest with hyperresonant lungs on percussion and dullness on percussion at the left base. Coarse rales and rhonchi are present at both bases.

Dr. Thakkar believed Mr. K. had left lower lobe pneumonia and acute exacerbation of COPD. Chest x-ray showed chronic changes with findings of pneumonia. Dr. Thakkar continued to list COPD secondary to coal workers' pneumoconiosis as a secondary diagnosis.

Dr. John A. Michos
(DX 2)

On October 12, 1999, Dr. Michos, board certified in pulmonary disease and internal medicine, reviewed numerous chest x-rays, Mr. K.'s treatment notes, and pulmonary evaluations by Dr. Castle and Dr. Morgan. Based on the preponderance of chest x-ray interpretations, Dr. Michos opined that Mr. K. had idiopathic pulmonary fibrosis and not coal workers' pneumoconiosis. Mr. K. was totally disabled from coal mine employment due to heart and lung disease. Dr. Michos recommended a CT scan "with high resolution" to help further identify the fibrosis and determine the presence of rounded opacities which would be indicative of simple coal workers' pneumoconiosis.

Dr. John R. Forehand
(DX 14)

On May 31, 2002, Dr. Forehand, board certified in allergy, immunology, and pediatrics,[45] conducted a Department of Labor-sponsored pulmonary evaluation of Mr. K. Dr. Forehand noted that Mr. K. worked in the coal mines underground for 37 years. He has a history of colds, pneumonia, wheezing, heart disease, and high blood pressure. He smoked half a pack of cigarettes per day from 1968 to 1988. Mr. K. presented with sputum, wheezing, dyspnea for 21 years, cough, and chest pain. A physical exam of the chest revealed crackles at the base. The x-ray was positive for coal workers' pneumoconiosis and arterial blood gas study showed hypoxemia at rest and with exercise. The pulmonary function test was normal.

Dr. Forehand concluded that Mr. K. has coal workers' pneumoconiosis based on the chest x-ray, arterial blood gas study, and physical exam, which was caused by his exposure to coal dust. Dr. Forehand also opined that Mr. K. had coronary artery disease that was caused by cigarette smoking and arteriosclerosis. He also believed that Mr. K. had a significant respiratory impairment based on the insufficiency of his residual oxygen transfer capacity. Therefore, Mr. K. cannot perform his last coal mining job and he is totally disabled from a pulmonary standpoint. Dr. Forehand concludes that coal workers' pneumoconiosis significantly contributes to Mr. K.'s respiratory impairment because of his coal mine employment history, physical findings, the appearance of the chest x-ray, and pattern of disability. Since Mr. K. does not have emphysema, the contribution from cigarettes to his respiratory impairment is not as great.

[45]I take judicial notice of Dr. Forehand's board certification and have attached the certification documentation.

Dr. James R. Castle
(DX 2, DX 23, and EX 6)

On October 18, 1995, Dr. Castle, board certified in pulmonary disease and internal medicine, conducted a comprehensive medical record review of Mr. K.'s records dating back to 1965. Dr. Castle supplemented his report in a deposition conducted on October 25, 1995. Mr. K. complained of a productive cough, shortness of breath, chest pain and orthopnea. Mr. K. worked in the coal mines for 35 years and performed a significant amount of heavy labor as a section foreman during his last years. He has a smoking history of 20 to 36 pack-years. His medical history includes angina and angioplasty and there is evidence of coronary artery disease.

The preponderance of the chest x-ray interpretations were positive for the presence of coal workers' pneumoconiosis. The valid pulmonary function tests completed by Mr. K. showed some obstruction and were diagnostic of a restriction but did not confirm the presence of these abnormalities. It appeared that the degree of restriction, which was mild, remained the same over time and Dr. Castle attributed the impairment to cigarettes. A significant reduction in Mr. K.'s diffusing capacity was also noted between 1983 and 1995, which Dr. Castle also related to tobacco smoke. Dr. Castle found that Mr. K. had a mild obstructive impairment and mild hypoxia related to empyema, which is a complication of pneumonia. Despite Mr. K.'s mild degree of respiratory impairment, Dr. Castle believed that Mr. K. could perform his previous coal mining job. Coal dust is not a factor in Mr. K.'s lung impairment because of the nature of his abnormalities.

After reviewing numerous chest x-rays, on November 29, 1995, Dr. Castle opined that the noted profusion of opacities were not indicative of coal workers' pneumoconiosis. Due to the irregular shape of the opacities, the location of irregular opacities solely in the lower lobes, and the notable absence of rounded opacities in the middle and upper lung zones, the radiographic opacities are not coal workers' pneumoconiosis or related to coal mine dust exposure. Dr. Castle also believed Mr. K. was totally disabled; however, the disability was not caused by coal workers' pneumoconiosis.

Dr. Castle conducted another pulmonary evaluation on March 15, 1999 of Mr. K. Mr. K. had trouble with shortness of breath since the 1970's. He could only walk 150 feet on level ground or up ½ a flight of stairs without stopping. He has a productive cough and sometimes wheezes. He has a history of pneumonia. He began smoking at age 25 and quit 10 years before the examination. While smoking, he smoked between a ½ pack and a pack of cigarettes per day, giving him about a 25 pack-year history. He worked in the mines for 36 years, ending his coal mine employment in 1983. As a section foreman, he did not perform a great deal of heavy labor but did a lot of different work.

A chest exam revealed a few early rales in both bases that cleared with heavy breathing. A chest x-ray did not show changes of coal workers' pneumoconiosis but there was evidence of bullae, cardiomegaly, emphysema, and lower zone fibrosis. Coal workers' pneumoconiosis does not cause lower zone fibrosis and thus the changes seen are most consistent with interstitial pneumonitis or bronchiectasis. Lung function tests showed an insignificant degree of obstruction which corrected with bronchodilation and no restriction. A moderate reduction in diffusing

capacity was also present. Dr. Castle concluded that there was no evidence of coal workers' pneumoconiosis based on radiographic evidence, physical examination, and lung function testing. There is evidence of bullous emphysema. Dr. Castle diagnosed chronic bronchitis induced by smoking, coronary artery disease, and angina pectoris. Dr. Castle believed the radiographic evidence indicated the presence of an interstitial process.

After a review of additional medical data, Dr. Castle concluded that Mr. K. does not have coal workers' pneumoconiosis. Mr. K. was exposed to coal dust in the coal mines for a sufficient time to develop the disease; however, his second risk factor, tobacco smoke, also could have caused a lung disease. Additionally, Mr. K.'s history of coronary artery disease and previous empyema could also have caused a restrictive lung disease. Mr. K.'s physical conditions were not consistent with coal workers' pneumoconiosis. The x-rays showed the presence of bullous emphysema, which is not caused by the inhalation of coal dust. Other changes on the x-ray are not consistent with coal workers' pneumoconiosis. Pulmonary function studies are not consistent with coal workers' pneumoconiosis as there is no evidence of a mixed irreversible obstructive and restrictive ventilatory impairment. Dr. Castle opined that Mr. K. could return to his previous coal mine employment as a section foreman. Even if it was determined that Mr. K. had pneumoconiosis, he is still not totally disabled from a pulmonary standpoint and does not have physiological changes from coal workers' pneumoconiosis. Moreover, if he does have pulmonary abnormalities sufficient to render him totally disabled, it would not be due to coal workers' pneumoconiosis. Any pulmonary impairment is due to the underlying tobacco smoke-induced bullous emphysema.

On June 26, 2002, Dr. Castle conducted a pulmonary evaluation of Mr. K. Mr. K. had been experiencing shortness of breath since before 1983 and was on oxygen at that time. Mr. K. becomes short of breath with bathing, showering, shaving and walking 25 to 30 feet on level ground or up a flight of stairs. Mr. K. also complained of a productive cough for several years. He sleeps on two to three pillows at night. He has a history of heart problems and had pneumonia in the past but has no asthma or TB (tuberculosis). Mr. K. reported a smoking history of half a pack to one pack of cigarettes per day for about 34 years, quitting 14 years earlier. He worked in the coal mines for 33 years, last working in 1983 when he was in a mine accident. As a section foreman, Mr. K. performed some heavy labor because he filled in for men working in the mine.

A physical exam of Mr. K.'s chest revealed that he was using accessory muscles with quiet breathing, percussion was noted, and crackles were present throughout both lung fields. The chest x-ray Dr. Castle interpreted was negative for pneumoconiosis but there was evidence of extensive bilateral interstitial fibrosis consistent with idiopathic pulmonary fibrosis. The pulmonary function test revealed a mild restrictive lung disease. Dr. Castle concluded based on his examination of Mr. K. that there was no definitive evidence of coal workers' pneumoconiosis, radiographic changes were consistent with idiopathic interstitial pulmonary fibrosis, mild restrictive lung disease with severe diffusion secondary to pulmonary fibrosis, coronary artery disease, and a history of pneumothoraces.

Dr. Castle conducted an additional medical record review at the time of his examination, wherein he reviewed medical reports and tests from Mr. K.'s earlier benefits claims and Dr.

Forehand's May 2002 report. After this additional review, Dr. Castle believed that Mr. K. did not have coal workers' pneumoconiosis. Mr. K. worked in or around the underground coal mine for a sufficient amount of time to have developed coal workers' pneumoconiosis, 33 years, however, Mr. K. was also exposed to an additional risk factor, cigarette smoke. Mr. K.'s history of smoking ½ a pack to one pack of cigarettes per day is sufficient to have caused COPD. Another risk factor that causes shortness of breath is cardiac disease. Additionally, Mr. K.'s x-ray changes were not consistent with pneumoconiosis because the x-ray distinctly showed extensive bilateral interstitial fibrosis primarily in the lower lung zones with honeycombing. These are not changes typical of coal workers' pneumoconiosis, which usually occurs with the presence of small, rounded, regular opacities. Idiopathic pulmonary fibrosis occurs with the presence of coarse, linear, irregular opacities, which may be associated with honeycombing. The condition is also associated with increased incidence of pneumothoraces. Therefore, radiographic changes are not consistent with pneumoconiosis.

The physiologic studies showed no significant large airway obstruction, which is against a diagnosis of both tobacco smoke-induced COPD and coal workers' pneumoconiosis. Mr. K.'s mild restrictive lung disease with a severe diffusion abnormality is consistent with idiopathic pulmonary fibrosis. Coal workers' pneumoconiosis does not typically cause a reduction in diffusing capacity but when it does, it is usually associated with p or r type opacities, which was not the finding in this case. The changes are physiologically consistent with idiopathic pulmonary fibrosis. Mr. K. does have a severe diffusion abnormality which has resulted in significant hypoxemia. The hypoxemia is further exacerbated with exercise, which is also consistent with idiopathic pulmonary fibrosis. Mr. K. is totally disabled to such an extent that he cannot perform his usual coal mining duties. This impairment is due to idiopathic pulmonary fibrosis and is not coal workers' pneumoconiosis. Mr. K. is also disabled by coronary artery disease, which is not related to his coal mining employment or coal dust exposure.

On May 25, 2004, Dr. Castle testified in a deposition about his findings regarding Mr. K.'s pulmonary condition after reviewing Dr. Rosenberg's April 2004 medical report, Dr. Wheeler's interpretation of the April 20, 2004 x-ray, and medical records from October 2003 through February 2004. He stated that the presence of crackles in Mr. K.'s lungs indicates the presence of interstitial pulmonary fibrosis. Mr. K.'s episodes of pneumothoraces from which Mr. K. suffers, which Dr. Castle explained was an occurrence of a collapsed lung, is also a complication associated with idiopathic pulmonary fibrosis. The pulmonary function and arterial blood gas tests showed a lung restriction and even more significantly that Mr. K.'s diffusion capacity is severely reduced, which is typical of the progression of a fibrotic process. Dr. Castle does not attribute Mr. K.'s smoking history to his pulmonary impairment because there is not airway obstruction, only restriction. Mr. K.'s impairment is from interstitial pulmonary fibrosis, not from COPD, smoking, or coal dust exposure. His pulmonary impairment renders him totally disabled and he is unable to return to his previous coal mining job, but his total disability is not related to his previous coal dust exposure. Dr. Castle does not think Mr. K. has legal or medical pneumoconiosis.

Dr. David Rosenberg
(EX 4 and EX 7)

On April 20, 2004, Dr. Rosenberg, board certified in pulmonary disease and internal medicine, conducted a pulmonary evaluation of Mr. K. and reviewed past medical records. Dr. Rosenberg noted Mr. K.'s report of worsening respiratory condition for the last several years. He previously had pneumonia, a pneumothorax, and breathing difficulties for 30 years. He is on chronic oxygen and complains of shortness of breath with activities of daily living, and coughing with sputum production. He sleeps on two pillows. Mr. K. reported smoking from his 20's until 1989, between ½ a pack and one pack of cigarettes per day. He worked in coal mine employment for 33 years, ending in 1983 when his breathing was getting worse. As a section foreman, he helped the men in different capacities with hands-on work.

A physical exam of Mr. K.'s chest revealed chronic rales at the bases with decreased breath sounds. Mr. K.'s breathing condition has worsened markedly in the last four to five months. He had a problem with a pneumothorax and recently had pneumonia. He also has coronary artery disease. Dr. Rosenberg concluded that Mr. K. "clearly has interstitial lung disease which is causing profound hypoxemia and severe impairment." The interstitial lung disease has a mid and lower lung zone location and a linear character, which is not the pattern of coal workers' pneumoconiosis. Coal workers' pneumoconiosis has an upper lobe predominance with a micronodular configuration and when it worsens, it causes mass formation and large densities develop. Mr. K. does not have the x-ray appearance of coal workers' pneumoconiosis.

Dr. Rosenberg believes "there is no question Mr. K. is impaired and totally disabled from a pulmonary perspective." He has marked hypoxia with moderate restriction and cannot perform his previous coal mining job. His disability results from his interstitial lung disease, which is not coal workers' pneumoconiosis but rather a condition such as idiopathic pulmonary fibrosis. Asbestosis can cause similar changes but Mr. K. has not had the occupational exposure. Mr. K. has a disabling interstitial lung disease, which is not coal workers' pneumoconiosis because the features of his clinical situation are consistent with idiopathic pulmonary fibrosis. This condition was not caused by the inhalation of coal dust.

In a deposition taken on June 1, 2004, Dr. Rosenberg further explained his conclusions regarding Mr. K.'s pulmonary condition. The spirometry study showed a moderate restriction without abstraction, which is consistent with idiopathic pulmonary fibrosis. The arterial blood gas results showing hypoxia and problems with Mr. K.'s diffusing capacity are also consistent with idiopathic pulmonary fibrosis. Between the time of Dr. Castle's June 2002 examination and Dr. Rosenberg's examination, Mr. K.'s pulmonary function markedly decreased. This rapid progression is also consistent with idiopathic pulmonary fibrosis. Dr. Rosenberg does not believe that Mr. K. has either legal or medical pneumoconiosis. His findings of "progression of low diffusing capacity and low oxygen over time in the setting of a progression of linear changes with a lower lobe configuration is not a condition which has been caused or hastened by coal dust exposure." The only way coal workers' pneumoconiosis could cause these findings would have been with the presence of progressive massive fibrosis, which could be seen on the x-ray.

Discussion

Over the course of 25 years, several physicians who evaluated and or treated Mr. K.'s pulmonary condition disagreed on whether he has pneumoconiosis.[46] In light of this conflict in medical opinion, I must assess the respective probative value of these diverse assessments in terms of documentation and reasoning.

As to the first factor, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor affecting relative probative value, reasoning, involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

With these two factors in mind, and due to the two types of pneumoconiosis, I will separately address the probative value of the physicians' opinions in terms of clinical (20 C.F.R. § 718.201(a)(1)) and legal (20 C.F.R. § 718.201(a)(2)) pneumoconiosis.

Clinical Pneumoconiosis

Dr. Hess, Dr. Abernathy, Dr. Sutherland, Dr. Morgan, Dr. Fino, Dr. Dahhan, Dr. Castle, Dr. Thakkar, Dr. Patel, Dr. Forehand, and Dr. Smiddy based their respective diagnosis of clinical pneumoconiosis on positive chest x-rays. However, I have determined that at best the preponderance of the 70 chest x-rays in Mr. K.'s record is inconclusive and does not support a diagnosis of pneumoconiosis. As result, these multiple diagnoses of clinical pneumoconiosis established by radiographic evidence have little probative weight since they are based on inaccurate documentation.

Consistent with the preponderance of the radiographic interpretations, Dr. Masri, Dr. Michos, and Dr. Rosenberg presented reasoned opinions based on accurate documentation that Mr. K. does not have clinical pneumoconiosis.

Accordingly, the preponderance of the probative medical opinion does not support a finding of clinical pneumoconiosis under 20 C.F.R. § 718.202(a)(4).

[46]During his hospitalization of Mr. K., Dr. Andrews did not express an opinion about the presence of pneumoconiosis.

Legal Pneumoconiosis

During the course of their treatment of Mr. K. or based upon pulmonary examinations, Dr. Hatfield, Dr. Sutherland, Dr. Patel, Dr. Edwards, Dr. Smiddy, Dr. Thakkar, and Dr. Forehand diagnosed coal workers' pneumoconiosis. At the same time, also based on treatment, examinations, and medical record reviews, Dr. Masri, Dr. Abernathy, Dr. Morgan, Dr. Fino, Dr. Dahhan, Dr. Castle, and Dr. Rosenberg concluded Mr. K. did not have coal workers' pneumoconiosis.[47]

Prior to considering the diverse medical opinion on the presence of legal pneumoconiosis, I note once again that 20 C.F.R. § 718.201(b) defines legal pneumoconiosis as any chronic pulmonary disease or impairment significantly related to coal mine dust exposure. As a result, the analysis on the presence of legal pneumoconiosis under 20 C.F.R. §718.202)(a)(4) is essentially an inquiry on whether the preponderance of the probative medical opinion establishes the presence of coal workers' pneumoconiosis based on other objective medical evidence beyond chest x-rays.

With the two probative value factors in mind, I first give Dr. Hatfield's 1979 diagnosis of "possible" coal workers' pneumoconiosis diminished probative value due to its equivocal nature. Additionally, due to the dated nature of his medical assessment, Dr. Hatfield was unaware of the medical evidence that developed over the course of the next 25 years.

Dr. Masri's diagnosis of asthmatic bronchitis and COPD (chronic obstructive pulmonary disease), apparently unrelated to coal mine employment has little probative value because his treatment notes provide no explanation on the aspects of his treatment and examination that led to his conclusion.

Although he noted the presence of clinical pneumoconiosis in chest x-rays, Dr. Abernathy nevertheless reached a reasoned opinion that Mr. K.'s pulmonary impairment was unrelated to his coal mine employment based on the nature of the oxygen tension during the arterial blood gas studies and Mr. K.'s obstructive small airways impairment. However, Dr. Abernathy's pulmonary diagnosis loses probative value due to incomplete documentation. Since Dr. Abernathy rendered his assessment in 1983 and 1984, he was unaware of the subsequent, and extensive, objective medical evidence related to Mr. K.'s pulmonary conditions.

Dr. Sutherland's May 1986 hospitalization diagnosis of severe coal workers' pneumoconiosis has diminished probative value due to both its dated nature and the absence of any explanation of his conclusion. Specifically, Dr. Sutherland did not discuss how the various medical tests conducted during his treatment of Mr. K.'s acute shortness of breath lead to his diagnosis.

Although Dr. Edwards was well positioned as Mr. K.'s treating physician to render a probative assessment, his terse statement that both of Mr. K.'s pulmonary risk factors, cigarette

[47]Dr. Michos did not present an opinion on the presence of legal coal workers' pneumoconiosis. Instead, he recommended a CT scan to further assess the etiology of the totally disabling pulmonary fibrosis.

smoking and coal mine employment, contributed to his COPD has little probative value in the absence of any explanation.

As another treating physician, Dr. Patel also had the opportunity to present a documented and reasoned opinion. Yet, in regards to legal coal workers' pneumoconiosis, Dr. Patel initially diagnosed "possible" pneumoconiosis. In addition to the absence of sufficient explanation for his finding, the equivocal nature of that diagnosis diminishes its probative value. Later, when Dr. Patel included a more definite diagnosis of "underlying coal workers' pneumoconiosis," he simply cited medical history as the basis. Without any further explanation, that subsequent finding of pneumoconiosis based on medical history also has little probative value.

In his assessments from 1988 to 1998, Dr. Fino diagnosed the presence of pneumoconiosis. However, based on additional medical records, including successful treatments of Mr. K.'s pulmonary difficulties, and noting the fixed impairment associated with coal workers' pneumoconiosis, Dr. Fino presented a reasoned opinion that Mr. K.'s breathing problems were not related to his coal mine employment. Additionally, he opined that Mr. K.'s clinical presentation and bronchitis were consistent with a pulmonary impairment attributable to cigarette smoking. Again, though certainly reasoned, in a manner similar to the other earlier physician evaluations, Dr. Fino's conclusion suffers a loss of probative value due to incomplete documentation. Since Dr. Fino last evaluated Mr. K. in 1998, he was unaware of the subsequent worsening of Mr. K.'s pulmonary condition which appears to have become fixed in nature.

In a reasoned medical opinion, Dr. Forehand based his diagnosis of legal coal workers' pneumoconiosis on an arterial blood gas study, which demonstrated that Mr. K. has hypoxemia at rest and with exercise, and a physical exam of his chest, which revealed crackles at the base. However, his opinion loses probative value because Dr. Forehand based his assessment on one pulmonary examination and did not review additional recent medical records, including the October 2003 CT scan. The absence of additional documentation is significant because Dr. Forehand opined Mr. K.'s cigarette smoking was not a contributing factor to his impairment since he did not have emphysema. However, other radiographic evidence, including the CT scan, indicates that Mr. K. may have emphysema.

Relying in part on a mild obstructive pulmonary impairment demonstrated by a pulmonary function study and moderate hypoxia, in addition to radiographic opacities which were not consistent with coal mine dust related pneumoconiosis, Dr. Dahhan reached a reasoned medical opinion that Mr. K. did not have coal workers' pneumoconiosis. Instead, Mr. K.'s pulmonary impairment stemmed from his cigarette smoking. Yet, since Dr. Dahhan based his diagnosis on one pulmonary examination conducted in 1995, his opinion loses probative value due to limited documentation. In particular, Dr. Dahhan was unaware of the increasing severity of Mr. K.'s pulmonary impairment and the development of a restrictive component.

Similarly, due to limited documentation, Dr. Morgan's conclusion that Mr. K. does not have coal workers' pneumoconiosis has diminished probative value. Based on his extensive review of the medical evidence through 1995, Dr. Morgan's presented a reasoned medical opinion explaining how he concluded Mr. K. had interstitial fibrosis and emphysema unrelated to his exposure to coal mine dust. However, the extent of his documentary review stopped in 1995.

Consequently, he was unaware of the next 12 years of Mr. K.'s pulmonary history and medical evidence, including the October 2003 CT scan, which he recommended.

In light of his more than 20 years of treatment of Mr. K., Dr. Thakkar's diagnosis of legal coal workers' pneumoconiosis rests on a substantial documentary foundation. However, his diagnosis of legal pneumoconiosis is not well reasoned for three reasons. First, although he concluded Mr. K.'s COPD was secondary to his coal workers' pneumoconiosis, Dr. Thakkar did not identify the objective medical tests or clinical presentation that supported his diagnosis. Second, in a later diagnosis of coal workers' pneumoconiosis, other than radiographic evidence (which I have determined inconclusive, rather than positive for pneumoconiosis), Dr. Thakkar only identified one other element supporting his finding – Mr. K.'s extensive history of coal mine employment, which standing alone is an insufficient basis for a diagnosis of coal workers' pneumoconiosis. Third, Dr. Thakkar also found Mr. K. had an emphysematous chest but failed to discuss how he determined Mr. K.'s deteriorated pulmonary condition was related to coal workers' pneumoconiosis rather than emphysema.

Although serving as another treating physician, Dr. Smiddy's medical opinion loses probative value because his legal pneumoconiosis diagnosis is based on inaccurate documentation and is not well reasoned. Initially, Dr. Smiddy diagnosed coal workers' pneumoconiosis but noted that a "broad different diagnosis would be possible" as well as "multiple concomitant diagnoses." Nevertheless, Dr. Smiddy continued to diagnose Mr. K. with coal workers' pneumoconiosis, principally based on his initial evaluation that the chest x-rays indicated the presence of pneumoconiosis. Yet, that interpretation is inconsistent with my finding that the radiographic evidence in the record does not establish the presence of pneumoconiosis. Thus, Dr. Smiddy's diagnosis is based on inadequate documentation. In his subsequent evaluations of Mr. K. and corresponding treatment notes, Dr. Smiddy continued to include in his diagnosis summary coal workers' pneumoconiosis, in addition to interstitial fibrosis, but failed to include any further explanation for his assessment.

The remaining physicians, Dr. Castle and Dr. Rosenberg, well qualified as board certified in pulmonary disease, presented well reasoned assessments based on extensive documentation. In terms of well integrated medical analysis, Dr. Castle and Dr. Rosenberg discussed nearly every aspect of the medical evidence in Mr. K.'s case to differentiate and isolate idiopathic pulmonary fibrosis, rather than pneumoconiosis due to coal dust or COPD due to cigarette smoke and coal dust, as the cause of Mr. K.'s impairment. Noting the clinical presentation of crackles, the location and nature of the radiographic opacities, the restrictive impairment coupled with a severe reduction in diffusion capacity, and the absence of micro-nodularities in the recent CT scan, Dr. Castle and Dr. Rosenberg presented the best reasoned conclusions that Mr. K.'s totally disabling pulmonary impairment is due to scarring caused by idiopathic pulmonary fibrosis unrelated to his long term exposure to coal dust.

Dr. Castle specifically opined that the chest x-rays and pulmonary function tests provided evidence that Mr. K. has idiopathic pulmonary fibrosis. Dr. Castle found that the x-rays showed extensive bilateral interstitial fibrosis consistent with idiopathic pulmonary fibrosis and the pulmonary function test revealed a mild restrictive lung disease with severe diffusion, which is caused by pulmonary fibrosis. He noted evidence of interstitial pulmonary fibrosis included the

presence of crackles in Mr. K.'s lungs and incidences of pneumothoraces. Dr. Castle explained that Mr. K.'s shortness of breath symptoms were caused by COPD, which developed from Mr. K.'s smoking history and cardiac disease.

Dr. Rosenberg also believes that Mr. K.'s pulmonary impairment is the result of his interstitial lung disease, which is a condition such as idiopathic pulmonary fibrosis and is not the result of coal dust inhalation. Dr. Rosenberg explained that Mr. K.'s spirometry study, showing a moderate restriction without obstruction, is consistent with idiopathic pulmonary fibrosis as is his arterial blood gas study, which revealed hypoxia and diffusing capacity abnormalities. Dr. Rosenberg additionally noted that the rapid decrease in Mr. K.'s pulmonary function between Dr. Castle's exam in June 2002 and his evaluation in April 2004 is consistent with idiopathic pulmonary fibrosis. Finally, Dr. Rosenberg explained that the only way coal workers' pneumoconiosis could have caused the findings in Mr. K. would have been with the presence of progressive massive fibrosis, which would have been observed on the x-rays.

In summary, for diverse documentation and reasoning issues all but two of the medical opinions have diminished probative value. The remaining two opinions by Dr. Castle and Dr. Rosenberg are well reasoned, documented, and most consistent with all the medical evidence in the record. Their probative medical consensus establishes that Mr. K. does not have legal coal workers' pneumoconiosis. Accordingly, I conclude Mr. K. cannot establish the presence of legal pneumoconiosis in his lungs through the preponderance of the more probative medical opinion, under 20 C.F.R. § 718.202(a)(4).

Compton Analysis

Under the guidance of the decision in *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), I must also consider the chest x-ray evidence and medical opinion together to determine whether Mr. K. has pneumoconiosis. In that regard, since standing alone neither the preponderance of the chest x-rays nor the preponderance of the probative medical opinion establish the presence of clinical or legal pneumoconiosis, consideration of that evidence together obviously still fails to produce a finding of pneumoconiosis.

Conclusion

On remand, the opinions by Dr. Castle and Dr. Rosenberg on the particular issue of the causation of his totally disabling pulmonary impairment have diminished probative value. As a result, through the remaining probative opinion of Dr. Smiddy, Mr. K. has established a change in condition by showing that his total disability was due to coal workers' pneumoconiosis. However, upon consideration of the entire record, I find that Mr. K. has not established the presence of pneumoconiosis through radiographic evidence or probative medical opinion. Having failed to prove this requisite element for entitlement to black lung disability benefits, Mr. K.'s subsequent claim for benefits must be denied.

ORDER

The black lung disability benefits claim of MR. C.K. is **DENIED**.

SO ORDERED:

A

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: June 22, 2007
Washington, DC

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).

Attachment No. 1

American Board of Medical Specialties
Certification:

Dilip B. Patel, MD

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Attachment No. 2

American Board of Medical Specialties
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Attachment No. 3

American Board of Medical Specialties
Certification:

John Randolph Forehand, MD

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